APPENDIX F POTENTIAL HEALTH EFFECTS FROM RELEASE OF NITROGEN DIOXIDE (NO_2)	

General Chemistry of Nitrogen Dioxide

 NO_2 is a gas above about 70 degrees F. It is reddish-brown. It is heavier than air, with a vapor density of 1.58 (air = 1.0). It has a pungent odor. The odor threshold, or concentration at which most people can identify it, is roughly 1-6 ppm. One part per million (ppm) in air = 1.88 mg/m³, equal to 1,880 μ g/m³.

Nitrogen dioxide is highly reactive. It is a strong oxidizing agent and is corrosive to metals. It combines with water to form nitric acid (HNO₃) and nitric oxide (NO).

Nitrogen dioxide is significantly different from nitrous oxide or dinitrogen monoxide, designated by the molecular formula N_2O and usually referred to as laughing gas. In contrast to nitrogen dioxide, nitrous oxide is rather stable, and although it is a central nervous system depressant and an asphyxiant, is not irritating to the respiratory tract. It is used as an anesthetic, especially in dentistry, at concentrations of up to 70% (700,000 ppm) with no respiratory toxicity.

Sources of NO₂ and NO

Nitrogen dioxide and nitrous oxides are formed from the incomplete combustion (burning) of nitrogen-containing chemicals. Incomplete combustion means that the combusted materials are not broken down completely. The concentrations of NO and NO₂ are expected to be about the same, or predominately NO₂, because NO reacts with atmospheric oxygen and chemically converts to NO₂ after both are formed. Sources of NO₂ include automobiles, welding, natural gas appliances, cigarette smoke, and explosives.

Toxicity

Nitrogen dioxide gas may cause significant toxicity because of its ability to form nitric acid with water in the eye, lung, mucous membranes and skin.

Nitrogen dioxide is considered to have poor "warning properties" because it is not immediately sensed at concentrations which can cause significant lung damage or even death.

The exact concentrations at which NO_2 will cause various health effects cannot be predicted with complete accuracy, because the effects are a function of air concentration and time of exposure, and precise measurements have not been made in association with human toxicity. The information that is available from human exposures also suggests that there is some variation in individual response. The concentrations in the following discussion are therefore approximate.

Acute Exposure

When inhaled NO₂ may cause death by acute broncho-spasm (airway closing), pulmonary edema (damage to fine tissues of the lung), or bronchiolitis obliterans (obstruction of the fine lung passages, secondary to tissue damage). Potentially fatal pulmonary edema can occur following minimal early symptoms. Symptoms usually occur within 1-2 hours of exposure, but may be delayed up to 72 hours.

The IDLH ("immediately dangerous to life and health") concentration is 20 ppm. Depending on the length of exposure, high concentrations of NO_2 may also cause pneumonia (generalized lung inflammation), and bronchiolitis (inflammation of the bronchioles). Recovery may be either complete or may involve some degree of impairment of pulmonary function. Acute exposure to high concentrations of NO_2 may cause serious eye irritation and damage.

Chronic Exposure

Chronic or repeated exposure to lower concentrations of NO_2 may exacerbate preexisting respiratory conditions, or increase the incidence of respiratory infections. For example, in one study of workers in a diesel bus garage, respiratory symptoms were elevated when concentrations of NO_2 were above 0.3 ppm.

Persons considered sensitive to NO₂ toxicity include children, persons with decreased ventilatory reserves (the elderly and persons with COPD - chronic obstructive pulmonary diseases, including asthma, bronchitis and emphysema), and persons who are at risk for developing infections, such as persons undergoing chemotherapy for malignancies, persons with acute leukemia and patients who have had bone marrow transplantations.

Numerous indoor air studies on the effects of nitrogen oxides on respiratory infections indicates that NO_2 exposure increases the risk of respiratory symptoms in children aged 5 to 12 years. Although it may not be possible to estimate a relationship between concentration and effects, one analysis of these studies suggested that for every increase of 0.015 ppm in 2-week average exposures to NO_2 there is an increased risk of 20% of respiratory symptoms and disease.

Asthmatics may be the group most susceptible to NO_2 because their airways are hyper-responsive to irritants. In controlled studies, asthmatics have experienced symptoms, but not changes in lung function at concentrations as low as 0.5 ppm for 2 hours. Their responsiveness to exercise or other irritants, however, has been shown to be enhanced by NO_2 exposures as low as 0.1 ppm.

NO₂ Levels of Interest (1 ppm = $1.88 \text{ mg/m}^3 = 1,880 \mu\text{g/m}^3$)

(1 ppm 1:00 mg/m 1;000 μg/m)		
	ppm	mg/m ³
EPA National Ambient Air Quality Standard (yearly average)		0.09
EPA Significant Harm Level (1-hour average)		3.76
Odor threshold		1.90-11.20
OSHA Permissible Exposure Level (PEL) ¹		5.64
OSHA Short-Term (15 min) Exposure Limit (STEL) ²		9.40
NIOSH recommended Short-Term Exposure Limit (15 min)		1.88
NIOSH Immediately Dangerous to Life & Health (IDLH) ³		37.60

NO₂ is a SARA TITLE III Extremely Hazardous Substance (40 CFR Table 302.4)

Reportable Quantity: 10 pounds

Threshold Planning Quantity: 100 pounds

PEL (Permissible Exposure Levels) are enforceable workplace standards which may not be exceeded during any 8-hour work shift of a 40-hour work week. In July 1992 the 11th Circuit Court of Appeals vacated more protective PELS set by OSHA in 1989, moving them back as standards set in 1971. NO₂ was changed back to 3 ppm from 1 ppm. In 1989 OSHA had adopted a ceiling (a value which should not be exceeded at any time) of 5 ppm. There is no current ceiling level for NO₂.

² A STEL is a 15-minute time-weighted average exposure that should not be exceeded at any time during a workday.

³ The IDLH is "a condition that poses a threat of exposure to airborne contaminants when that exposure is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such environment." (NIOSH, 1994).